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**The risk of Eating Disorders comorbid with Attention-Deficit /
Hyperactivity Disorder: A Systematic Review and Meta-Analysis**

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RUNNING HEAD: ADHD COMORBID WITH EATING DISORDERS REVIEW

Abstract

Objective: There has been interest in whether people with Attention-Deficit/Hyperactivity Disorder (ADHD) are at higher risk of developing an Eating Disorder (ED). The aim of this study was estimate the size of this association with a meta-analysis of studies. **Methods:** We retrieved studies following PRISMA guidelines from a broad range of databases. **Results:** Twelve studies fitted our primary aim in investigating ED in ADHD populations (ADHD = 4.013/Controls = 29.404), and 5 exploring ADHD in ED populations (ED = 10.44/Controls = 11.292). The pooled odds ratio of diagnosing any ED in ADHD was increased significantly, 3.82 (95% CI:2.34-6.24). A similar level of risk was found across all ED syndromes [Anorexia Nervosa = 4.28 (95%CI:2.24-8.16); Bulimia Nervosa = 5.71 (95%CI: 3.56-9.16) and Binge Eating Disorder = 4.13 (95%CI: 3-5.67)].The risk was significantly higher if ADHD was diagnosed using a clinical interview [5.89 (95%CI:4.32-8.04)] rather than a self-report instrument [2.23(1.23-4.03)]. The pooled odds ratio of diagnosing ADHD in participants with ED was significantly increased, 2.57 (95%CI:1.30-5.11). Subgroup analysis of cohorts with binge eating only yielded a risk of 5.77 (95%CI:2.35-14.18). None of the variables examined in meta-regression procedures explained the variance in effect size between studies. **Discussion:** People with ADHD have a higher risk of comorbidity with an ED and people with an ED also have higher levels of comorbidity with ADHD. Future studies should address if patients with this comorbidity have a different prognosis, course and treatment response when compared to patients with either disorder alone.

The risk of Eating Disorders comorbid with Attention-Deficit / Hyperactivity Disorder: A Systematic Review and Meta-Analysis

A proportion of people with Eating Disorders (ED) have poor treatment outcomes (1). Identifying subgroups of patients with comorbid disorders may help explain differences in disease trajectories (2) and aid the process of tailoring treatments to match individual needs.

There has been interest in whether developmental disorders might increase ED risk. For example, Anorexia Nervosa (AN), especially the restrictive subtype, has been linked to autism (3) and the purging subtype of AN has been associated with ADHD (4). The association of Bulimia Nervosa (BN) and Binge Eating Disorder (BED) with ADHD has been repeatedly demonstrated (5,6). We reported a higher prevalence of ED, in particular BED, in a lean ADHD cohort in 2004 (6). This result was further replicated in 6 samples (4,7–11). A systematic review of all studies found that BN was the most frequent form of ED seen among people with ADHD (12). The frequency of ADHD symptoms is higher in BN or BED when compared to AN (13). We have published the first systematic review about ADHD comorbid with ED in 2008 (12) but this field of research was still in its infancy limiting us to perform a qualitative review of studies. Thus, there is the need to extend the previous systematic research and complement it with a meta analysis of studies to measure the risk of the association between ADHD and Eating Disorders.

The studies of ADHD patients found that the prevalence of BN ranged from 9 (7) up to 11% (14), whereas that of BED ranged from 9.3 (15) up to

11.4% (11), and that of AN was 1% (10). Conversely, when the prevalence of ADHD was investigated in ED, studies in AN patients found rates ranging from 3 (16) up to 16.2% (17), while for BN patients it ranged from 9 (16) up 34.9% (17), and for BED patients it was 19.8% (17). The positive association of ADHD and ED traits has also been demonstrated in correlational studies (13,18–24), with a correlation coefficient ranging from 0.23 (19) to 0.59 (22). The number of ADHD symptoms correlate with ED symptom severity in all binge/purge ED subtypes (13). ADHD symptoms have been found to predict binge eating severity (20,21,25) and bulimic symptoms even after controlling for anxiety and depression (19,25).

In the paediatric population, the symptom of Loss of Control Eating (LOC), a sub-clinical form of binge eating is also associated with ADHD rather than the full BN and BED syndromes probably because the two latter only evolve in the context of greater autonomy in food choice and consumption (26).

Although the most likely trajectory is for ADHD symptoms to precede the development of an ED it is possible that secondary consequences of an eating disorder on attentional and impulsive behaviours (27) lead to *de novo* ADHD traits, and to an ADHD-like syndrome.

The link mediating the association of ADHD and ED is still not clear and is possibly a combination of genetic and environmental mechanisms. Genetic variability in dopaminergic genes related to reward processing have been described in a patient with comorbid ADHD and BED (28), in a case control study of obese BED comorbid with ADHD (29) and in BN women with a childhood history of ADHD (30).

Anomalies in attentional and impulsive mechanisms could explain the link between ADHD and ED. Impulsive behaviours constitute a core ADHD symptom domain (31) and deficits in its regulation have been demonstrated in this population (32). Also, impulsivity plays a role in binge eating and purging behaviours (33). Negative urgency, an aspect of impulsivity, that has been shown to relate binge eating to emotional regulation (34) could also play a role in explaining this association. Patients with ADHD+ED have higher levels of impulsive traits than patients with either of those conditions alone (22,25) .

Changes in different aspects of attentional functioning have been reported in ED. Patients with AN, BN and BED have deficits in both sustained attention and processing speed (27,35). The disturbed reward encoding in ED patients increases salience towards food cues. This leads to the development of attentional bias towards food and an altered motivational control (36). Programs that train attention towards neutral stimuli are beneficial in correcting these disturbances(37,38). ADHD individuals typically have motivational difficulties since they prefer immediate and small rewards instead of delaying gratification to obtain larger rewards(39). In concert with this style, ADHD individuals would be vulnerable for developing binge eating and would have difficulties in maintaining long lasting healthy habits. It is unfortunate that the presence of ADHD as a possible mediator explaining attentional deficits in ED has not yet been considered in the neuropsychological or attentional bias studies.

In terms of treatment, the recent approval of psychostimulants for the treatment of Binge Eating Disorder (BED) (40) raises the question as to whether improvements in an undiagnosed Attention-Deficit / Hyperactivity Disorder

(ADHD) syndrome could lead to additional benefits to the ED symptoms . Several case reports have described that patients with refractory Bulimia Nervosa (BN), associated with either a current or a past history of ADHD, improved their ED when treated with psychostimulants (41–46). However, in a recent lysdexamphetamine trial for BED, even excluding ADHD participants, significant improvements in eating behaviour were observed. It is well documented that psychostimulants can promote appetite loss and weight loss (47). Thus, it is a clinical concern that the use of psychostimulants could improve ED symptoms largely by a primary effect on appetite. In this case, the use of psychostimulants to treat ADHD in individuals with an undiagnosed ED could lead to severe weight loss (48), specially in impulsive individuals with risk of psychostimulant misuse (49).

The aims of this study was to update and expand the previous systematic review and perform a meta-analysis of studies to investigate the risk of comorbidity with an ED (AN, BN or BED) in ADHD individuals or eating disorder symptoms (binge eating or LOC eating) in ADHD paediatric samples. The risk of diagnosing ADHD in ED patients was also investigated. Furthermore, we explored the strength of association between ADHD and ED symptoms when those were measured dimensionally. Additionally, we explored confounding factors, which might explain the variability in ED prevalence in this population.

METHODS

The systematic review and meta-analysis were conducted following PRISMA statement guidelines (Preferred Reporting Items for Systematic Reviews and Meta-Analysis) (50). Selection procedures are presented in **Figure 1**. The following key terms were used: (“Eating Disorders”; “Anorexia Nervosa”; “Bulimia”; “Bulimia Nervosa”; “Binge Eating Disorder”; “Binge Eating”; “Overeating”) AND (“Hyperactivity with attention-deficit disorder”; “ADHD”; “ADD”; “Inattention”), to search for articles in PubMed, Scielo, PsychINFO and ISI Web of Knowledge databases.

Studies were searched independently by both the first (BPN) and second (CB) authors and were considered eligible if published in English, French, German, Dutch, Spanish or Portuguese languages; if they were of a case-control design; if they reported measures to calculate odds ratio; and if they had been peer-reviewed for publishing. Studies that did not specify how ADHD or ED was diagnosed were excluded. A hand-search from reference lists of selected articles was performed to find additional relevant studies. Finally, the lists of selected articles were compared and discordant references discussed. If both authors failed to reach a consensus this was later discussed with both principal authors (PM and JT). Finally, references were added to EndNote where duplicates were removed. All case-control studies characteristics are summarized in **Table 1**, while correlational studies are summarized in **Table 2**.

Statistical analysis

The eligibility criteria for inclusion in meta-analytic procedures required that studies reported either: a) the ED frequency in an ADHD versus a Control group, b) the ED frequency in an ADHD versus a Control group, c) a correlation coefficient between ADHD and ED symptoms. Moreover, authors were contacted in order to obtain unpublished quantitative data. Thus, the data used on Mikami et. al., 2008 (51); Mikami et. al., 2010 (52); Docet et. al., 2012 (53); Reinblatt et. al., 2015 (26); Reinblatt et. al., 2015 (b) (54), Sonnevile et. al., 2015 and Rojo-Moreno et al., 2015 (55) were kindly provided after direct contact with authors. The database from Nazar et. al., 2014 (25) was available for direct extraction of necessary outcomes. Additionally, after contacting one of the authors (Kessler RC) from the Collaborative Psychiatric Epidemiological Surveys (CPES), we used the available information from the National Comorbidity Survey – Replication (NCS-R, 2001-2003) dataset to extract results relevant for the purpose of the present analysis (56).

The Odds Ratio of having an ED amongst ADHD versus Control groups, and the odds ratio of having ADHD amongst ED versus Control groups was calculated using a random effects model. STATA 12 (Stata corporation, College Station, TX, USA) was used for this purpose as well as building all graphics. The user-written commands for STATA *Metan*, *Metabias* and *Metares* were used.

The pooled correlation coefficient among ED and ADHD symptoms measured in different rating scales was obtained by using the transformed value on a standardized Fisher's Z scale with the formula ($Z = \frac{1}{2} \ln(1 + r/1 - r)$) and it's standard error by ($SE_z = 1/\sqrt{n-3}$) (57). This meta-analytic model used the

Hedges-Olkin random-effects model. To calculate the correlation coefficient back to a non-standardized scale the formula $(e^{((2 \times Z_{\text{pool}} - 1) / (2 \times Z_{\text{pool}} + 1))})$ was used.

Heterogeneity across studies was calculated using Cochran's Q statistics, expressed in a chi-square result. Inconsistency across studies was investigated through publication bias with I^2 statistics $[(Q - df) / Q]$ (58).

The assessment of publication bias was done with the Harbord's test of bias, which was chosen for exploring "small studies effect" explaining the OR variance, as it is better suited for binary data and results in less false positive outcomes than the Eggers' test (59). Also, informal visual inspections of funnel plots were performed.

All studies were assessed with the case control version of the Newcastle-Ottawa Quality Assessment Tool (60). Quality assessments were used to investigate risk of bias. Each study could receive at least 4 points in the selection, 2 points in the comparability and three points in the exposure subscale.

Metaregression was used to investigate if mean age; weight category; proportion of females; sample size; method for ADHD diagnosis; method for ED diagnoses, study country or study quality would account for differences in studies results.

RESULTS

The risk of Eating Disorders in people with ADHD versus Controls

Outcomes for 33417 individuals (ADHD = 4013 / Controls = 29404), across 12 retained studies were available. Most paediatric studies measured symptoms such as LOC (26), binge eating episodes (54,61) or restrictive behaviours (23), whereas all adult studies reported on syndromes (AN, BN and BED). Two studies were considered outliers (54,62) since their effect size had a wide confidence interval that had an upper bound that was much bigger than all others (54,62), and were excluded from the analysis. Results including these two studies are presented in the online **Supplementary Materials**. All of the studies demonstrated higher levels of either ED diagnosis or disordered eating behaviours in the ADHD group compared with the control group.

The meta-analysis from pooling all 12 studies (9,10,25,26,51–53,56,61,63), after the exclusion of the two outliers, yielded a significant risk ($p<.001$) of diagnosing either an ED syndrome (AN, BN or BED) or ED symptoms (restrictive eating, binge eating episodes or LOC) within ADHD individuals, with an overall random effect OR of 3.81 (95% CI: 2.31 - 6.21) (**Figure 2**). The results of the key subgroup meta-analyses, heterogeneity tests and publication bias, are presented in **Table 3**.

To disentangle the individual risk for each ED, analyses were carried out for studies that reported AN (either full syndrome (10,64) or AN-related symptoms (23)), BN (all studies reported full syndrome (9,10,25,64)) and BED (either full syndrome (25,53,64) or LOC (26)). A separate analysis with studies that reported outcomes for binge eating episodes that fulfilled DSM criteria (Objective Binge Eating), regardless of the ED syndrome (9,10,25,53,64), was also performed. The subgroup analysis yielded a risk of 4.28 (95% CI: 2.24-8.16)

for AN (n=3); 5.71 (95% CI: 3.56-9.16) for BN (n=4); 4.13 (95%CI: 3-5.67) for BED (n=4) and of 4.67 (3.58-6.10) for objective binge eating episodes (n=5), in ADHD.

In adults (n= 5) (9,25,53,63,64) the OR for ED syndromes was 4.09 (95% CI: 2.32 – 7.20). The subgroup analysis in the paediatric samples, also found an increased significant (p = .013) risk (n= 7) (10,23,26,52,61,65,66), OR of 3.59 (95% CI: 1.51 – 8.50) (**Figure 3**). The subgroup analysis in the obese only samples, also found an increased risk in the obese-ADHD (n=3 studies) (25,26,53), OR of 5.81 (95% CI: 3.15-10.7).

In the subgroup analysis by gender, females only (n=9) (9,10,23,25,26,52,61,63,66) presented an OR of 3.46 (95% CI: 2.00 – 5.98). Adult females (n= 3) (9,25,63) had a similar sized risk OR of 4.35 (1.67 – 11.27). In the paediatric female subgroup (n=6) (10,23,26,51,52,61) the risk for any ED was 2.96 (95% CI: 1.32-6.65). The subgroup consisting only of males (n=6) (9,23,26,52,61,63), had an OR of 3.37 (1.46 – 7.77).

Since studies differed in how they diagnosed ADHD we analysed results for subgroups that did so by using a semi-structured interview or by using questionnaires. The analysis of ADHD diagnosis by interview (n=8) (9,10,25,26,51,52,64,65) yielded a significant risk of ED, 5.89 (95% CI: 4.32 – 8.04), with a subset for adults only (n=3) (9,25,64) of 5.80 (95%CI: 4.04 – 8.25) and a subset for paediatric only (n=5) (10,26,51,52,65) of 6.22 (95% CI: 3.20 – 12.11). These results significantly differed from those that diagnosed ADHD through questionnaires (n=4) (23,53,61,63), which resulted in a significant risk of 2.23 (95% CI: 1.23 – 4.03).

The overall analysis resulted in substantial inconsistency across studies (**Table 2**). Thus, we performed a meta regression analysis to investigate whether age group (paediatric or adult), BMI class (obese only or all weight categories), percentage of female participants, country where research was conducted, study quality rating, method for diagnosing ED and method for diagnosing ADHD (self-report or clinical interview) might have contributed to the findings. Although these variables resulted in an adjusted R^2 of 63.64%, this was not significant ($p = 0.49$).

The risk of ADHD in people with Eating Disorders versus Controls

A total of 12,336 individuals (ED = 1044 / Controls 11,292), across 5 retained studies were available. Again, two studies (22,25) were excluded for their effect size, which again, had a wide confidence interval that had an upper bound that was much bigger than all others. Results including these two studies are presented in the online **Supplementary Materials**. Most studies (26,55,67) found higher rates of ADHD in ED populations, although the results did not reach formal levels of significance in two (68,69). As in the ADHD studies, not all authors reported which type of ED syndrome was comorbid with ADHD in their samples (**Table 1**). There were not enough observations to run a model of meta regression.

The combined risk of diagnosing ADHD in the five ED studies was significant ($p = .007$), 2.57 (95% CI: 1.30 – 5.11) (**Table 3**). The analysis of the adult subgroup ($n=3$) (67–69) resulted in a non-significant risk of 1.58 (95% CI: .78-3.19).

A subgroup analysis of patients with binge eating (68) (including BED (67) or LOC only (26) patients), produced (n=3) (26,67,68) a significant risk of , 5.77 (95% CI: 2.35-14.18).

The strength of association between ADHD and ED symptoms

A pooled correlation coefficient of $r = .43$ (95% CI: .33 – .52). was found from the association of ADHD and ED symptoms in seven studies (13,19–22,25,70) The subgroup using bulimic symptom questionnaires (n=4) (13,19,25,70) yielded a pooled correlation coefficient of .38 (95% CI: .28 - .49), while the subgroup using binge eating questionnaires (n=3) (20,21,25) resulted in a pooled r of .47 (95% CI: .31 - .59).

DISCUSSION

The aim of this study was to provide an update on a previous systematic review of the literature examining the comorbidity between ADHD and ED (12) and to perform a meta-analysis. To the best of our knowledge this is the first meta-analysis investigating the risk of the ADHD+ED comorbidity.

The risk of ADHD individuals having an ED is increased three-fold and the risk of ED individuals also having ADHD is increased two-fold. The strength of association between ADHD and ED symptoms is moderate. No variables in our meta-regression explained the effect size variance. The sample division into clinically relevant subgroups failed to show that size of the potential risk differed significantly. For example, the risk of ED in males and females were both

significant with overlapping confidence intervals. The size of the increased risk for disordered eating in paediatric samples was similar to the risk of eating disorders in adult samples. The results from the meta analysis suggested that the risk for all ED syndromes in ADHD is similar, with a non-significant trend for a higher risk of BN.

The significantly higher risk of ADHD when the diagnosis was made by using a semi-structured interview suggests that the association with ADHD is robust. Most studies didn't report if collateral information was used to define ADHD cases. Collateral reports are suggested as best practice for making adult ADHD diagnosis (71), however, agreement rates between adults and their parents range from moderate (71) to high (72). Finally, adult patients diagnosed with ADHD by self-report whose informants did not report ADHD symptoms in childhood have similar clinical profiles and treatment response as those who did (73).

Most ADHD symptom domains have been found to be associated with ED behaviours. Both inattention and impulsivity predicts bulimic symptoms (22,25,52,66), while both inattention and hyperactivity predicts craving (26,61) (61). Of note, the most recent DSM edition (DSM-5)(74) has deemphasized former ADHD subtypes because of the recent literature on low stability rates of subtypes along time (75,76). Another possibility is that ADHD symptoms may be associated with an increased risk for ED with disturbances in body perception (77) as Fernandez-Aranda et al., 2013, demonstrated that problems in interoceptive awareness correlate with ADHD symptoms.

Interestingly, it has been suggested that attentional processes could dominate the physiological responses to self-image exposure in BN patients because of an abnormal arousal reaction with negative feelings when viewing their own bodies, probably due to an attentional bias (78).

It is noteworthy that the risk for the association between ADHD with ED is more than the double that found between obesity & ADHD. In two recent reviews on the subject, the risk of obesity in people with ADHD ranged from 1.37 (79) up to 1.55 (80) in adults when compared to controls, whereas in children it ranged from 1.13 (79) to 1.22 (80). Obese people with ADHD differ from those without ADHD as they have a higher frequency of disturbed eating behaviours (5,25) and Eating Disorders (ED), and more psychiatric comorbidities, suggesting that the mechanism underpinning this association is binge eating. Obesity has been found to be a long term consequence of BED in epidemiological studies (81) and one study has demonstrated that LOC mediates BMI increments in ADHD children (26). Further longitudinal studies tracking disordered eating and other confounding features such as medication from childhood into adult life and weight gain are of interest.

The Reward Deficiency Syndrome is a concept that links genetic abnormalities in dopaminergic genes to behavioural phenotypes. It is possible that this syndrome could be a transdiagnostic feature between ADHD (82), ED, Obesity (83) and Substance Abuse Disorders (84). As yet to our knowledge there are no genome wide association studies (GWAS) studies for BN and BED available to examine correlations in polygenic risk scores between these

conditions. The analysis of specific genetic profiles may shed light to understanding the occurrence of diagnostic subgroups within a disorder.

In terms of functional magnetic resonance imaging studies an overlap in circuit anomalies in response to a variety of cognitive and emotional regulation tasks have been found by Seymour et al., 2015. Interestingly, a dissociation occurred regarding reward processing studies as ADHD participants demonstrated a weaker neural recruitment during reward anticipation and a higher than expected neural recruitment during reward receipt (85), while, binge eaters had a hyperresponsivity to food-related stimuli in reward areas during anticipation and receipt (86). There are still no studies demonstrating if the brain functioning of patients with ADHD+ED differs from patients with either of these disorders alone.

Although different studies found that ADHD+ED patients had higher impulsive traits measured by questionnaires than patients with either of those disorders alone (20,22,24,25), these results were not replicated with the use of neuropsychological tests. The mediation between ADHD and binge eating wasn't explained by results from a Go/No-Go task (20). Also, there were no significant differences when comparing bulimics with or without a childhood history of ADHD in tasks measuring executive functioning (22).

The aforementioned explanations for the ADHD+ED comorbidity rely on single factors (e.g. impulsivity, genetic abnormalities) that do not account for the complexity of behaviours exhibited by both disorders. Hypothesis taking into account dual-process or even a three-level conceptualisation might convey a better understanding of this comorbidity. Strack & Deutsch (87) have proposed a

dual process integrating reflective and impulsive functioning as determinants of habit. It could be possible that a distorted system of cognitions and beliefs (ED) would interact with an altered response to environmental stimuli (ADHD), leading to the activation of an altered behavioural schemata. Sergeant, 2000 (88) conceptualised the cognitive energetic model for ADHD, with a first level of computational processing of attention depending on a second level of state factors (effort, arousal and physiological activation), managed by a third level of executive functioning. Using this model as a basis, we suggest that not only ADHD neuropsychological functioning could alter information processing for food-related decision making but also changes in nutritional or emotional state promoted by ED could make ADHD individuals more prone to disinhibition.

Limitations

The number of relevant studies is limited. Among the 12 case-control of ADHD versus Controls studies exploring this comorbidity, 5 focused on paediatric populations with an age range from 10 up to 17 years. The definitions of ED in paediatric studies were broad, and included LOC (26), disordered eating behaviours (51,54) in addition to DSM-5 ED syndromes. As the median age of treatment seeking for BN is 18 and for BED is 22 (89) it is probable that these paediatric cohorts will not have passed the age of maximum risk for ED. This might explain why these diagnoses were infrequent in paediatric populations and were absent in two studies (51,52). Also, few studies explored ADHD in ED versus Controls, which limited us from exploring those results with a meta regression. Given that recently lisdexamphetamine has been authorised to treat

BED (40) it would be interesting to know if the prevalence of ED is reduced in the context of this treatment. However there was insufficient data on psychostimulant use to add it as a covariate in the meta regression.

CONCLUSION

In the present meta-analysis, we have demonstrated evidence that ADHD individuals are at risk of disordered eating in childhood and eating disorders (AN, BN and BED) later in development. This research raises the question of whether children with ADHD should be screened for disordered eating and if so what form of intervention (psychological or pharmacological) might be used to moderate the risk.

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